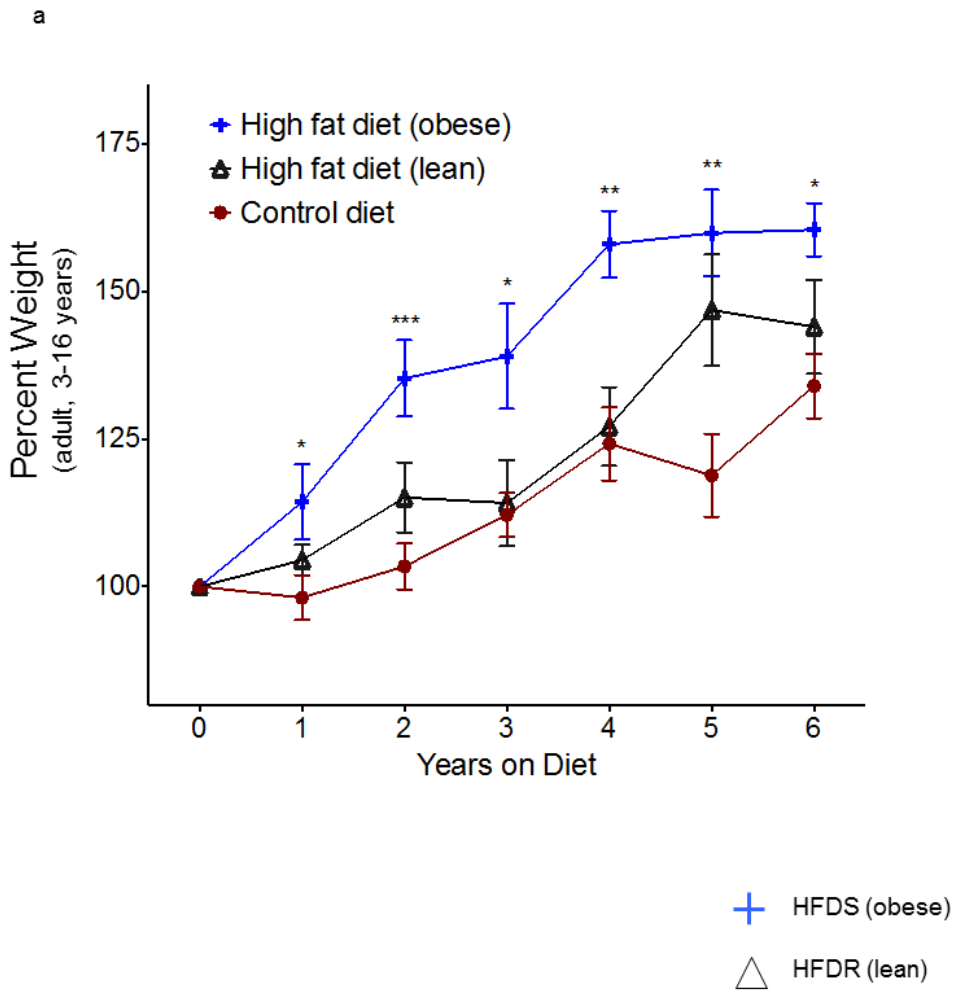
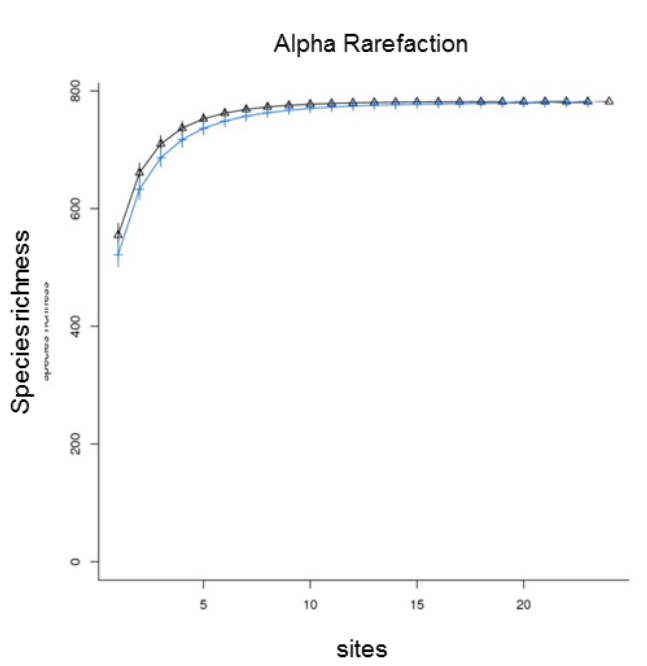
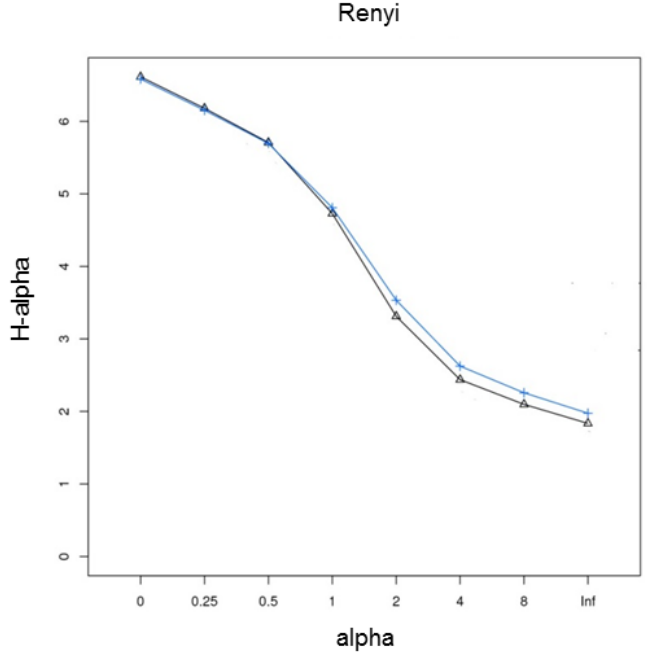




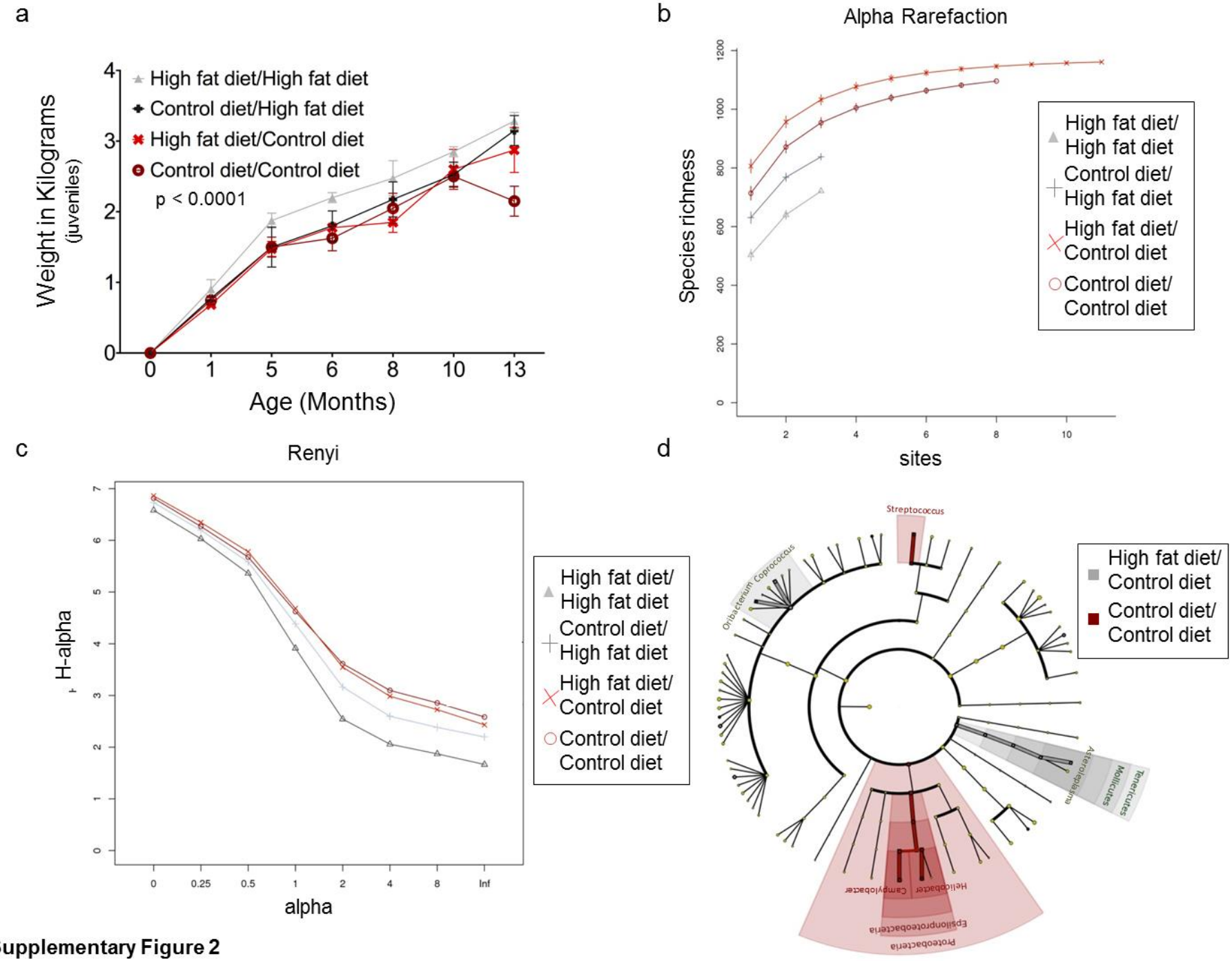


HFDS (obese)

HFDR (lean)

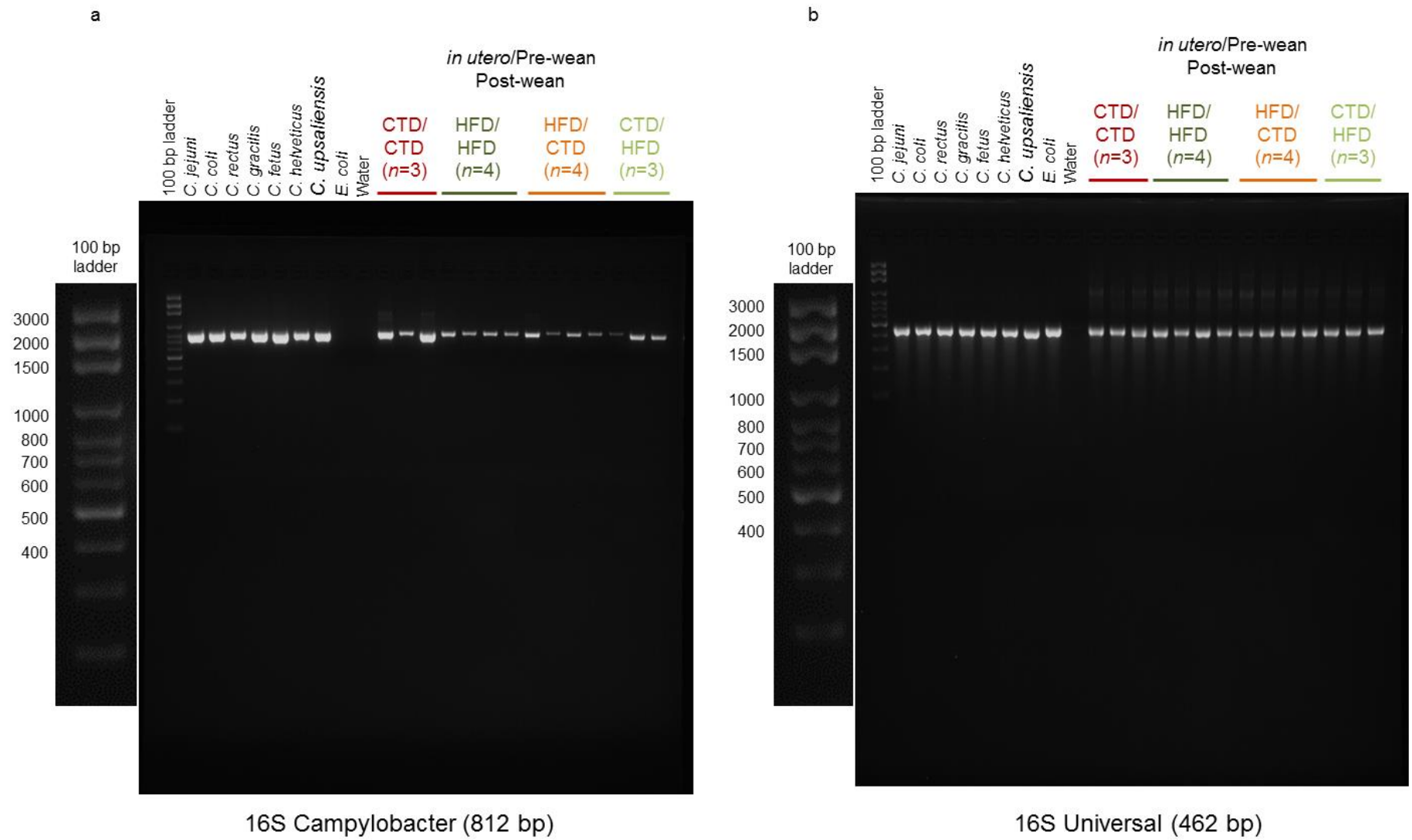
Supplementary Figure 1

Supplementary Figure 1: Diet, not habitus, influences changes in the maternal microbiome. *M fuscata* were maintained on a control or high fat diet. Intestinal samples were collected from either obese (HFDS) or lean (HFDR) *M. fuscata* maintained on an isocaloric diet consisting of 13% fat (control diet) or 36% fat (high fat diet). DNA was isolated from intestinal samples and 16S pyrosequencing (454 FLXtitanium) was performed. Sequences were analyzed using QIIME software. (a) Percent weight increase was calculated by dividing the weight of an animal at years 1-6 on the diet by their weight at the initiation of the diet. Closed red circles indicate dams on a control diet (lean), black open triangles indicate dams individuals on a high fat diet (lean), and blue crosses indicate dams on a high fat diet (obese). n=14 animals per group. Statistical analysis was performed using a one-way ANOVA with error bars indicating standard error of the mean (SEM). Statistical significance is indicated between control diet (lean) and high fat diet (obese) individuals. * p<0.05 **p<0.005 ***p<0.0005 (b) Graph depicting the species richness calculated by rarefaction of lean (open triangles) or obese (blue crosses) animals consuming a high fat diet. n =15 animals per group. (c) Renyi plot of alpha diversity of lean (open triangles) or obese (blue crosses) animals consuming a high fat diet. n =15 animals per group.



Supplementary Figure 2

Supplementary Figure 2: Diversity of juvenile *M. fuscata* microbiota is affected by *in utero* diet and post-weaning diet. *M. fuscata* were vaginally birthed to mothers consuming a control or high fat diet. Infants consumed the maternal diet until weaning when they were either maintained on the maternal diet (control cohort) or switched to the opposing diet (crossover cohort). Cohorts were determined based on maternal/post-wean diets. Red open circles designate juveniles exposed to a control diet/control diet, closed gray triangles designate juveniles exposed to a high fat diet/high fat diet, red crosses designate juveniles exposed to a high fat diet/control diet, and black crosses designate juveniles exposed to a control diet/high fat diet. (a) Weight was measured at various time points during the first year prior to euthanasia. While weight did not appear to vary significantly between cohorts at the time of measurement, weight gain between the cohorts did vary significantly ($p < 0.0001$). Statistical analysis for weights at the time of measure was performed using a one-way ANOVA, and statistical analysis for weight gain was analyzed by two-way ANOVA of repeated measures ($p < 0.0001$). $n = 2$ juveniles per group. (b) Graph depicting the species richness of intestinal samples was calculated by rarefaction of juvenile cohorts. (c) Renyi plot depicting the alpha diversity of intestinal samples from juvenile cohorts. (d) Cladogram obtained by LEfSe analysis. Shaded red areas indicate bacterial taxa with higher abundance in intestinal samples from juveniles exposed to control diet/control diet while gray shaded areas indicate bacterial taxa with higher abundance in intestinal samples from juveniles exposed to a high fat diet/control diet. (b-d) $n = 3$ juveniles exposed to control/control diet, $n=2$ juveniles exposed to high fat diet/high fat diet, $n=4$ juveniles exposed to high fat diet/control diet, and $n=3$ for juveniles exposed to control diet/high fat diet.



Supplementary Figure 3

Supplementary Figure 3: Exposure to a high fat diet diminishes the presence of *Campylobacter*. *M. fuscata* were vaginally birthed to mothers consuming a control or high fat diet. Infants consumed the maternal diet until weaning when they were either maintained on the maternal diet (control cohort) or switched to the opposing diet (crossover cohort). At one year of age, animals were sacrificed and DNA was isolated from the stool. DNA was subjected to PCR amplification for both universal 16S rRNA and for *Campylobacter* 16S rRNA genes. The number of juveniles (*n*) in each cohort is indicated in parenthesis in the figure legend. (a) PCR amplification of *Campylobacter* 16S rDNA (812 bp) and (b) universal 16S rDNA (462 bp). PCR products were run on a 3% agarose gel containing ethidium bromide.